

Severe fetal tachycardia after administration of hexoprenaline to the mother

A case report

T. M. D'HOOGHE, H. J. ODENDAAL

Summary

A fetus developed extreme supraventricular tachycardia (210/min) after intravenous hexoprenaline 10 µg was administered to the mother for intrapartum fetal distress. Urgent caesarean section resulted in the birth of a male baby with a reduced Apgar score; the infant survived after being ventilated for 24 hours.

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Case report

A 19-year-old primigravida was admitted to hospital for preterm labour, when the gestational age of the fetus was 32 weeks. The fetal heart rate (FHR) was 158/min. Since the cervix was 4 cm dilated and contractions were fairly strong, both indomethacin and hexoprenaline were used for tocolysis. Glucocorticoids were administered on admission and again 12 hours later to enhance fetal lung maturity. Since the uterine contractions were easily suppressed, the intravenous hexoprenaline infusion was stopped after 4 hours and replaced with fenoterol 5 mg orally every 4 hours.

Three days later the patient developed a urinary tract infection. The temperature rose to 38°C and abdominal tenderness was present. Since intra-uterine infection was likely, it was decided to discontinue the fenoterol treatment and to rupture the membranes. Oxytocin infused at a rate of 1 mU/min and later at a rate of 2 mU/min was sufficient to stimulate adequate contractions. Intravenous antibiotics were also started. Internal monitoring of the FHR demonstrated a tachycardia of 160 - 175/min and then a sudden deceleration to 80/min without clear clinical or cardiotocographical evidence of hypertonic uterine contractions. The oxytocin was immediately discontinued and hexoprenaline 10 µg was given intravenously (Fig. 1). The FHR rapidly increased to at least 210/min.

Since delivery was not imminent, a caesarean section was performed. The Apgar scores of the male infant were 2,3 and 5 after 1,5 and 10 minutes, respectively; the heart rate never decreased below 100/min. The birth weight was 1440 g and the neonatal assessment of the gestational age was 37 weeks.

The newborn infant was admitted to the intensive care unit where he was ventilated for 24 hours. Indications for ventilation were the low Apgar score and the possibility of infection. However, both aerobic and anaerobic cultures from the amniotic fluid and placental membranes were negative. The first blood gas values after admission to the intensive care unit were as follows: pH 7,31; bicarbonate 22 mmol/l; carbon dioxide pressure 4,8 mmol/l; and oxygen pressure 23 mmol/l. There was no suggestion of long-lasting asphyxia. Recovery was uneventful and no cardiac abnormalities could be detected.

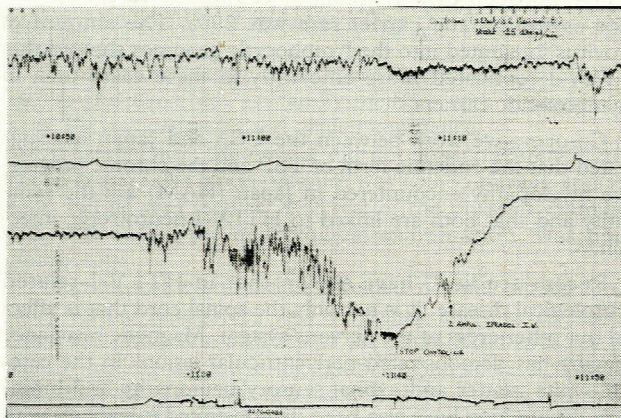


Fig. 1. Fetal heart rate of at least 210/min after administration of intravenous hexoprenaline to the mother.

Discussion

In this case β -sympathomimetic drugs combined with maternal fever and fetal distress could have caused the severe fetal tachycardia of at least 210/min. In a prospective study on intrapartum fetal tachycardia in 30 patients, Hager and Pauly¹ found that fetal tachycardia was a reliable indicator of maternal and neonatal infective morbidity. In their study, 43% of the mothers had infants with an intrapartum FHR of more than 180/min. However, an FHR of more than 200/min, as in this case, was not mentioned. Lipshitz *et al.*² showed that hexoprenaline tocolysis for intrapartum fetal distress and acidosis does not have any significant maternal or fetal side-effects. Recently, β -sympathomimetic drugs (including ritodrine, fenoterol, terbutaline but not hexoprenaline) were reported to have the following fetal and neonatal complications: paroxysmal supraventricular tachycardia; atrial flutter; myocardial infarction; cardiac failure; pulmonary oedema; hydrops fetalis; and perinatal death.³ According to Maxwell *et al.*⁴ each instance of fetal tachycardia needs to be considered as a cause of non-immune hydrops fetalis. It is unlikely, however, that congenital tachyarrhythmia existed in this case because the FHR preceding the period of tachycardia and following the delivery was normal and fetal hydropic changes were absent. In our

Department of Obstetrics and Gynaecology, University of Stellenbosch and Tygerberg Hospital, Parowvallei, CP
T. M. D'HOOGHE, M.D. (BELGIUM) (Present address: Universitaire Ziekenhuizen, Louvain, Belgium)
H. J. ODENDAAL, M.B. CH.B., F.R.C.O.G. (S.A.), M.MED. (O. & G.), M.D., F.R.C.O.G.

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Reprint requests to: Professor H. J. Odendaal, Dept of Obstetrics and Gynaecology, PO Box 19063, Tygerberg, 7505 RSA.

case the supraventricular tachycardia was probably caused by the β -sympathomimetic drugs and aggravated by the underlying tachycardia due to infection. The mechanism involved is probably prolonged and elevated activation of the adrenergic receptors.

To the best of our knowledge, this is the first case report of an adverse fetal reaction to hexoprenaline tocolysis for intra-partum fetal distress.

REFERENCES

1. Hager WD, Pauly TH. Fetal tachycardia as an indicator of maternal and neonatal morbidity. *Obstet Gynecol* 1985; **66**: 191-194.
2. Lipshitz J, Shayer DC, Anderson GD. Hexoprenaline tocolysis for intra-partum fetal distress and acidosis. *J Reprod Med* 1986; **31**: 1023-1026.
3. Katz VL, Seeds JW. Fetal and neonatal cardiovascular complications from β -sympathomimetic therapy for tocolysis. *Am J Obstet Gynecol* 1989; **161**: 1-4.
4. Maxwell DJ, Crawford DC, Curry PV, Tynan MJ, Allan LD. Obstetric importance, diagnosis, and management of fetal tachycardia. *Br Med J* 1988; **297**: 107-110.

Haemostasis by angiographic embolisation in exsanguinating haemorrhage from facial arteries

A report of 2 cases

T. MOKOENA, A. T. O. ABDOOL-CARRIM

Summary

Life-threatening exsanguinating haemorrhage from arteries of the face following trauma is uncommon. When it occurs it is often located in the relatively inaccessible parts of the vessels and requires deep face or neck exploration and ligation of the main feeding vessel. The procedure requires expert head and neck vascular surgery performed under general anaesthesia, which is often not suitable in these haemodynamically unstable patients. In addition, surgery is often rendered more difficult by the associated post-traumatic swelling and disfigurement. Because of these considerations, angiographic embolisation of the bleeding vessels was performed as an alternative to surgical exploration. This report illustrates its use in achieving haemostasis in 2 patients.

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Exsanguinating haemorrhage as a result of trauma to arteries of the face is uncommon.^{1,2} When it occurs, it usually involves the most inaccessible parts of the afflicted vessel. Under these circumstances surgical exploration of the face and ligation of the bleeding vessel has been the accepted practice.^{1,3} It is sometimes more expedient to ligate the ipsilateral external carotid artery,^{1,4} which forms the main trunk of arterial supply to the face. This obviates extensive dissection of the face, which would be necessary to identify and ligate the specific bleeding vessel, in an already haemodynamically unstable patient. Neck and face explorations are attended by significant morbidity, especially when undertaken by relatively inexperienced

surgeons and under emergency conditions. Moreover, the swelling and distortion of the anatomy of the face that often accompanies trauma compounds the difficulty of this surgery. It was felt therefore that surgical exploration was not altogether satisfactory and examination of other avenues for induction of haemostasis was undertaken. Angiographic embolisation has been used successfully at our institution to achieve haemostasis in bleeding from renal arteries,⁵ branches of the coeliac axis,^{6,7} and bronchial arteries. It was therefore decided to try this manoeuvre in the setting of exsanguinating haemorrhage of the arteries of the face.

The first 2 patients with facial injuries who were successfully managed by angiographic embolisation to achieve haemostasis are described.

Case reports

Case 1

A 24-year-old man presented at the casualty department of King Edward VIII Hospital in a state of circulatory shock following a single stab wound in the left submandibular area. The haemoglobin level was 6.4 g/dl on arrival. Following resuscitation with intravenous crystalloid fluids and blood transfusion, the patient began to 'vomit' and to regurgitate copious amounts of fresh blood.

Because there were no immediate operating facilities available to explore and ligate the bleeding vessel surgically, it was decided to try an angiographic embolisation manoeuvre. Temporary haemostasis was secured by manual external compression of the ipsilateral common carotid artery. A transfemoral left external carotid angiogram demonstrated a bleeding left lingual artery, which was embolised with a coil spring, and haemostasis secured. The procedure was performed under local anaesthetic and light sedation in an otherwise conscious patient. Total blood loss was in excess of 3 l before haemostasis was finally achieved, and the patient

Department of Surgery, University of Natal and King Edward VIII Hospital, Durban

T. MOKOENA, D. PHIL., F.R.C.S.

A. T. O. ABDOOL-CARRIM, F.R.C.S.

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